within two days. On the tenth day, the patient complained of sore throat. The tonsils were red with small patches of white exudate on them. Another 10 cc. of gamma globulin was given. By the twelfth day, the throat had cleared and the patient felt well. He remained asymptomatic thereafter, was discharged on the 33rd day and returned to work two weeks later. Results of liver function tests had returned to normal by the time he returned to work. However, electrocardiograms—the most recent one (Figure 3) was made May 2, 1957—continued to show complete left bundle branch block.

SUMMARY

A case of infectious mononucleosis is presented in which complete left bundle branch block developed. The bundle branch block persisted after the patient clinically recovered. This is believed to be the first reported case of this complication.

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Intramedullary Hemangioma of the Spinal Cord

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THE OCCURRENCE of spinal cord hemangiomas limited to the intramedullary site is rare.[6] A recent report of nine cases of vascular malformations of the spinal cord[1] included two cases of intramedullary lesions and seven in which there were both intramedullary and extramedullary lesions. In 1926 Foix and Alajouanine[5] described progressive spinal cord destruction associated with intramedullary and extramedullary vessel abnormalities consisting of an increase in the number of vessels as well as thickening of the walls. No vascular occlusions or thrombi were noted. They termed this spinal cord involvement "subacute necrotic myelitis." Wyburn-Mason[9] said that subacute necrotic myelitis is a form of angioma racemosum venosum in which vessel changes in the substance of the cord are the primary lesion and vessel changes externally may not be evident.

In reviewing reports of these vascular malformations of the cord[1,2,5,6,7,8,9] the following characteristics were noted: Onset in middle age, predominantly in men; variable pains preceding the onset of paraplegia; paraplegia usually of the spastic type but progressing to flaccidity unless the lesion is focal; sensory involvement, which occurs later than paresis, is at first dissociated but becomes complete; frequently elevation of cerebrospinal fluid protein without pleocytosis; the course is usually subacute, with death occurring in one to two years; diagnosis of the lesion is possible if it is not limited to an intramedullary position, the myelographic appearance consisting of serpentine rarefactions in the column of Pantopaque.[3]

Treatment of vascular malformations of the cord has been successful only when the lesion is at least partially in an extramedullary position, with improvement occurring following surgical decompression but with no response to x-radiation.[4]

The pathologic changes are usually grossly apparent. There is an increase in the number of both arteries and veins and often they are tortuous and dilated. The vessels may be entirely extramedullary, more rarely intramedullary alone and most commonly both within and without the cord substance. The abnormal vessels may be extensively distributed, the entire cord being involved in some cases. Less commonly the lesion may be relatively focal—limited to one or two segments. On section the vessels may have thickened walls, some with almost complete obliteration of the lumen. Occasionally there are organized thrombi. Necrosis and malacia appears in the cord within the distribution of the vascular lesion, and at times there is syrinx formation.

From the Veterans Administration Hospital, Oakland.
Submitted December 12, 1956.

REPORT OF A CASE

A 58-year-old white man was admitted to the Veterans Administration Hospital, Oakland, for the first time on August 30, 1955, because of aching pain in the left upper quadrant of the abdomen for about two weeks. The pain was constant, was not related to meals and was not associated with nausea or vomiting. A few days after onset, the pain moved to the right side, then into the spine and gradually extended up between the shoulder blades and down into both arms. Two days before admission the patient noted weakness of the left leg and difficulty in urinating. There was no history of trauma.

The patient appeared chronically ill and was barely able to walk; the gait was spastic. Upon neurological examination the only sign of cranial nerve abnormality was that the pupils were somewhat sluggish in reacting to light. The upper extremities appeared to have normal strength, coordination and tone. Both lower extremities were weak, the left more so than the right. Deep tendon reflexes were hyperactive in all extremities. Ankle clonus was not elicited. Superficial abdominal reflexes were absent. Babinski's sign was present bilaterally. Pronounced tenderness to touch and pressure was noted over the right upper chest anteriorly but the sensitive area was not well demarcated. Pinprick perception was impaired below the eighth thoracic level on the right side.

No abnormalities were noted in the blood or urine on the day of admittance to hospital. A serologic test was negative for syphilis. The cerebrospinal fluid on the day following admission was faintly xanthochromic and clear. The pressure of the cerebrospinal fluid was 60 mm. of water. No evidence of blocking was evoked by the Queckenstedt maneuver. The fluid contained 5 lymphocytes per cu. mm. Total protein content was 56 mg. per 100 cc. The gold curve was 1122100000. The result of a Kolmer test was negative for syphilis.

In an x-ray film of the chest minimal pulmonary emphysema and slight increased density in the hilar areas were noted. X-ray films of the thoracic spine showed minimal scoliosis to the right, centered at the seventh vertebra. Slight hypertrophic fringing of the vertebral bodies was noted.

The patient's condition became worse rapidly. Within a week after admission there was complete loss of sensation below the second thoracic dermatome on the right and the eighth thoracic dermatome on the left. Both legs were completely paralyzed and were flaccid. Bladder control was completely absent. The patient continued to complain of severe pain in the right upper chest as well as in the left shoulder and arm. A myelogram four days after admission showed no evidence of block or defect in the opaque medium. Two weeks after admission the left hand was quite weak and sensory deficit became apparent in both arms up to the seventh cervical dermatome. An electroencephalogram tracing was "borderline"; there was some diffuse 5 to 7 per second activity, particularly over the frontal lobes. No abnormalities were noted in x-ray films of the skull.

The patient began to complain of deafness and upon evaluation a partial perceptive deafness bilaterally was noted.

Two months after admission, moderate hematemesis occurred and a Sippy diet and antacid drugs were prescribed. No evidence of an active ulcer was noted in roentgen studies, but there was deformity and irregularity of the duodenal area suggesting scar formation at the site of an old ulcer. Despite nursing care to prevent it, decubitus ulceration of the right hip developed four months after admission. A month later urinary tract infection occurred. It was resistant to all antibiotics.

Results of spinal fluid examination in January of 1956 showed it to be completely within normal limits. Urinary obstruction and urinary extravasation occurred, and suprapubic cystotomy and surgical incision and drainage were carried out. At that time the blood urea nitrogen was 19 mg. per 100 cc. The patient was febrile from then on and was confused and restless. On February 24, 1956, he became unresponsive and comatose. The next day he died.

Autopsy

At autopsy moderate aortic arteriosclerosis in the abdominal region was noted. There was pronounced erosion of the distal half of the esophageal mucosa, and a small abscess was present in the right kidney. Decubitus ulcers were present over both hips, the sacrum and the right ilium. Bronchopneumonia and congestion were noted in microscopic examination of lung tissue. The spleen showed pronounced hyalinization and subintimal thickening of smaller arterioles. In one of the adrenal glands a small vein was distended by a thrombus, with early organization.

In the brain there was some 20 cc. of clotted dark red blood in the subarachnoid space at the tip of the left frontal lobe and about 15 cc. of blood in the floor of the right middle fossa as well as about 10 cc. between the dura and arachnoid overlying the right temporoparietal lobes. The circle of Willis had no left posterior communicating artery. The

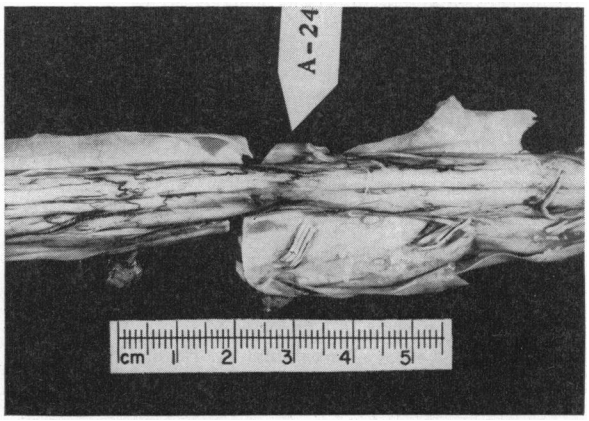

Figure 1.—Gross specimen of spinal cord showing narrowing at the fourth thoracic level.

brain weighed 1250 gm. Some medial degeneration of arterioles in the region of the internal capsule, with the deposition of a blue-black pigment in the media was observed in histologic examination. One small cyst-like area was noted near the cerebral cortex but involving the white matter, with the nearby surrounding vessels showing thickened and eosinophilic walls.

In the spinal cord a 1.5 cm. area of malacia was observed at the level of the fourth and fifth thoracic nerve roots. In this region the cord was 0.8 cm. in greatest diameter, while above and below this level it was 1.2 cm. (Figure 1). The surface of the cord was yellowish-brown in the narrowed area. A branch of the anterior spinal artery was distended and filled with blackish, old-appearing clotted blood at this level. At the fourth thoracic level the normal structure of the cord was almost completely obliterated by remaining necrotic cord substance and blood. As high as the sixth cervical segment there was discoloration and cavitation in the region of the central gray matter, more pronounced on the right side. Below the level of the fourth thoracic segment no gross changes were seen. Upon microscopic examination, finely vacuolated areas were observed in the posterior and lateral columns above the level of the lesion. A section (Figure 2) through the central portion of the primary lesion (at the fourth thoracic segment) showed diffuse degeneration of the normal cord tissue with a large cystic area which had an endothelial lining apparent in part of its margin. Numerous dilated blood vessels were present, some with irregularly thickened walls. Within two intramedullary blood vessels there were thrombi, one of which was undergoing organization (Figure 3). The thrombi did not completely occlude the vessels. No evidence of neoplastic or proliferative vascular activity was seen. The leptomeninges about this portion of the cord were decidedly thickened, although the vessels within the leptomeninges were not greatly changed. In sections of the cord above the lesion, extension of an area of degeneration upward into the cervical region was noted. There a small cyst-like cavitation was noted, surrounded by gliosis. Degeneration of the posterior columns, of spinocerebellar and spinothalamic tracts above the lesion and of the corticospinal tracts below the lesion was observed in myelin-stained and fat-stained sections.

* * *

The case reported could not be diagnosed with certainty on clinical grounds as being one of spinal cord hemangioma, although the neurological symptoms were like those previously reported in this condition. Differentiation from an intramedullary neoplastic or focal infectious lesion would have required exploration. Occlusion of an anterior spinal artery would produce a similar syndrome. Myelography was not helpful, since no extramedullary vessel malformation was present. The blood noted in intracranial spaces at autopsy remained unexplained.

Presumably the symptoms in this condition came about from a failure of the abnormal vessels to supply the circulatory demands of the spinal cord. The thickening of the vessels may be a progressive degenerative atheromatous change taking place locally within a system of abnormal vessels where the flow of blood has always been sluggish. Or it may be that this change occurs only in persons in whom generalized arteriosclerosis develops, the congenital malformation in the spinal cord being a site of least resistance to such degenerative vascular processes. The fact that neurologic symptoms are delayed in appearance until middle age while the essential lesion is a developmental anomaly is compatible with either of these theories.

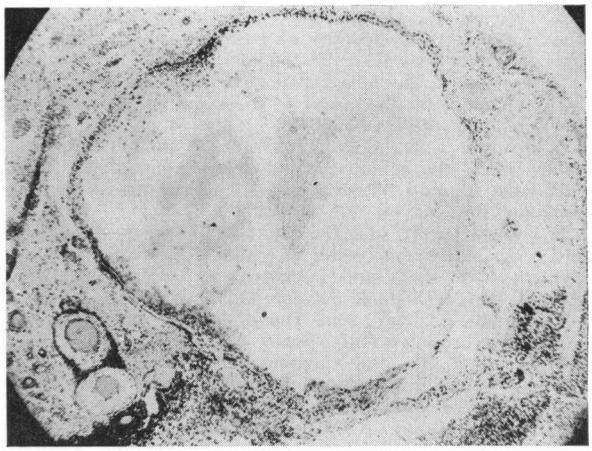

Figure 2.—Magnification of the cystic area at the fourth thoracic level. Its appearance, with an endothelial lining, suggests a dilated vascular channel. Two large vessels with thickened walls are present (Nissl stain, ×30).

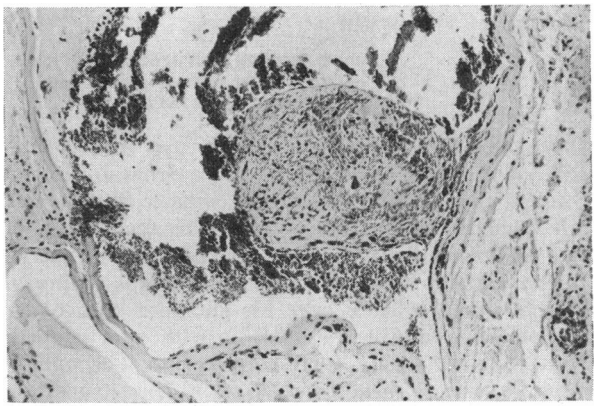

Figure 3.—Organized thrombus in a dilated blood vessel (Hematoxylin and eosin stain, ×100).

SUMMARY

A case of an intramedullary hemangioma with thickened and thrombotic vessels producing severe myelomalacia is reported.

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Migration of Bullet in Gunshot Wound of the Brain

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IN THE CASE here reported a bullet fired from a rifle entered a boy's head in the right frontal region, coursed through the brain and came to rest in the left parietal region just beneath the skull, then in five weeks gradually returned to the point of entry.

The patient, a boy 8 years of age, was riding a bicycle, looking back over his shoulder at a companion who, running to catch up with him, tripped and fell, discharging a .22 caliber rifle. The bullet, after traveling a distance of 40 or 50 feet, struck the patient in the right forehead just below the hairline and he tumbled from the bicycle. He was semiconscious when picked up by his mother some 20 or 30 minutes later. About an hour after the accident the patient, stretched out on the back seat of an automobile, was observed by one of the authors. In a state of moderate shock, he was pallid and sweating and the pulse was weak. The patient said he had heard a shot and had fallen from the bicycle.

He was taken to the hospital for treatment, and evidence of shock subsided. Neurological examination was done several hours later and the boy then was conscious and rational. Reflexes were equal and active and the Babinski response was not evoked.

X-ray films of the skull showed the wound of entry in the right frontal region and the major portion of the bullet resting in the left parietal area, just under the bone. Fragments of the bullet were distributed from the point of entry to the point at which the major portion of the bullet came to rest. Fragments of the bone were present in the right frontal area adjacent to the point of entry.

Antibiotic therapy was administered and in the ensuing few hours no evidence of progressive neurological involvement developed. Early the following morning the wound of entry was opened and debrided. Devitalized brain tissue and a few fragments of bone and metal were removed. Then the wound was closed. Antibiotic and anticonvulsant therapy was administered and the patient recovered. There was no sign of neurological impairment and the patient was discharged from the hospital.

Submitted January 24, 1957.

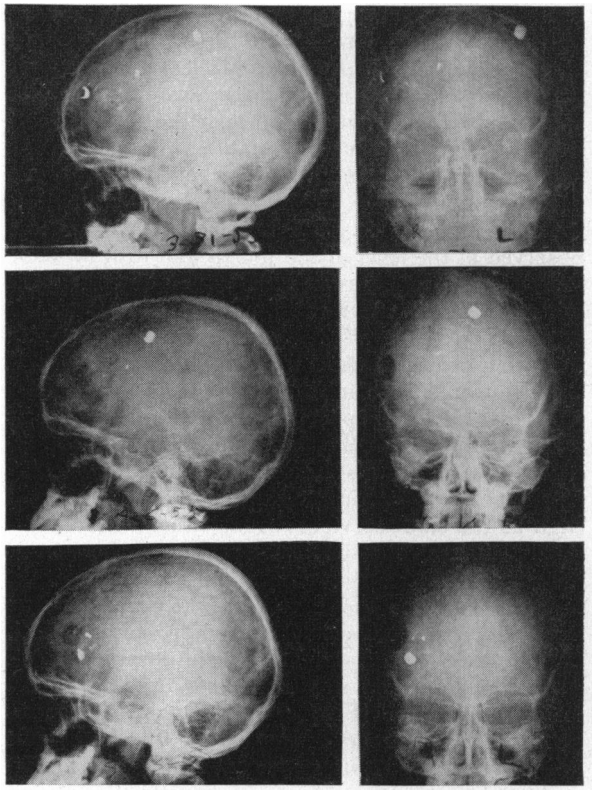

Figure 1.—*Top row:* X-ray films on day of accident, showing fragments of lead along course of bullet from point of entry in right forehead to point at which major portion came to rest against skull on left side. *Center:* Films taken a week later, after fragments of slug had been removed, showing main body of bullet returned part way along course of penetration. *Lower:* Five weeks after the accident, bullet almost back to point of entry.

Anticonvulsant therapy was continued. X-ray films taken at intervals thereafter showed the bullet to be migrating back toward the point of entrance. Although no attempt was made to keep the patient prone while asleep, he was instructed to lie in the prone position while awake and to jar his head gently against the bedding from time to time. Approximately five weeks after the accident, the bullet had returned almost to the point at which it had entered. Thereupon the main portion and a fragment of the bullet and several fragments of bone were removed from a point just inside the skull.

In the next year and a half no evidence of residual injury to the brain developed. No abnormalities were noted upon examination and the parents reported no changes in disposition or in personality. The patient returned to school, where he adjusted to classroom routine and appeared to have no loss of retentiveness.

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